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RECENTLY PUBLISHED SOVIET RESEARCH ON TOXICOLOGY

"Method of Detecting Arsenious Acid," M. M. Rudnev, P. M. Rudnev

"Zavodskaya Lab" Vol 13, 1947, p 128

To 3 ml of the solution neutralized to a pH of 7-8, 0.1N $AgNO_3$ is added dropwise to complete precipitation and then boiled for 3-4 minutes. Blackening of the precipitate after the boiling confirms arsenite. Phosphates, arsenates, sulfates, nitrates, chlorides, and starch do not interfere. Method for analysis of seeds treated with arsenious compounds suggested.

"Toxicity of Hexachlorocyclohexane," V. I. Vashkov, E. K. Serebryakova

"Med Parazitol i Parazitar Bolezni" Vol 16, No 1, 1947, pp 41-3

Mortality rates in white mice given different doses of hexachlorocyclohexane as an aerosol in starch paste and in apricot oil are presented. Effect of subcutaneous injections is also discussed; apparent toxic dose for guinea pigs is mentioned. Preparations do not seem to have any cumulative effect.

"Effect of Bandage Impregnated with Hexachlorocyclohexane Preparations on Rabbit," V. I. Vashkov

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"Med Parazitol i Parazitar Bolezni" Vol 16, No 1, 1947, p 435

Up to 10% solutions in dichloroethane or oil and up to 5% in kerosene were nontoxic; a 10% solution in the latter solvent was toxic.

"Crystal Structure of Cyanides: V, The Unit Cell and the Space Group of $K_4Fe(CN)_6.3H_2O$ Crystals (Tetragonal Type)," G. S. Zhdanov, V. A. Pospelov

"Zhur Fiz Khim" Vol 21, 1947, pp 521-2

In addition to pseudotetragonal crystals consisting of two normal monoclinic crystals, $K_4Fe(CN)_6$ forms on crystallization from H_2O tetragonal crystals, the unit cell of which contains eight molecules and has a = 9.35, c = 33.63 A. The lattice is body-centered and belongs to the space group $C^6_{4h} - J4_1/a$.

"Chronic Chloronitrobenzene Poisoning: I, Morphological Changes in the Blood; II, Morphological Changes of Internal Organs and the Central Nervous System," S. M. Dubashinskaya

"Farmakol i Toksikol" Vol 10, No 2, 1947, pp 51-8

Dogs were poisoned with the eutectic mixture of ortho- and para-chloronitrobenzene (I) by 4-6 hours daily exposure to air containing I vapor. Hemoglobin, leucocyte, erythrocyte, reticulocyte, normoblast, and methemoglobin counts were made before, during, and after poisoning. Anemia was a symptom, but there was also some activation of blood replenishment. There was some leucopenia in the first series. Morphological examination of marrow revealed increased hemopoiesis. Blood replenishment after chronic poisoning was slow. Under the same exposure conditions dogs were examined for effects on internal organs and effects on the central nervous system. Observed changes included atelectasis of pulmonary parenchyma; emphysema and atelectasis with expansion of pulmonary connective tissue; hyperplasia of Kupfer's cells; fatty infiltration, vascular leucocyte thrombosis and vacuole degeneration in liver cells; cardiac hypertrophy and fatty infiltration; chronic nephritis and subacute nephrosis. No significant gastro-intestinal or endocrine changes were observed. In the central nervous system there was some hyperplasia of cell structures, with hyperemia and vascular stasis. There were also changes in cerebral cortex and tissue.

"Significance of Sternal Puncture in Occupational Poisoning," E. I. Velling

"Farmakol i Toksikol" Vol 9, No 5, 1946, pp 50-2

Acute and chronic poisoning with TNT, N oxides, Cl, [illegible]ides, and fuel gas (propane-butane-butene) had effects which were studied in about 600 spinal puncture tests with rabbits and guinea pigs. Observations of spinal fluid were correlated with blood counts (erythrocytes, leucocytes, myelocytes, reticulocytes and lymphocytes, normoblasts, hemoglobin, eosinophilic and polychromatophilic cells). Examination of spinal fluid yields useful diagnostic and prognostic information.

"Pyruvic Acid Content of Blood in Tetraethyl Lead Poisoning," E. I. Veiling, A. A. Preobrazhenskaya

"Farmakol i Toksikol" Vol 9, No 5, 1946, pp 48-9

Vitamin B_1 helps to counteract excessive pyruvic acid (I) content in the blood of rabbits poisoned with $PbEt_4$. Assays of I in blood have diagnostic significance in $PbEt_4$ poisoning, and are useful for checking results of therapy. Increase of I in the blood after giving $PbEt_4$ is attributed to inhibition of coenzyme activity.

"Fluorine Poisoning in Farm Animals," A. P. Levenshtein, G. D. Dubrovin

"Veterinariya" Vol 23, No 4, 1946, pp 33-6

Description of symptoms and of the usual means of poisoning by F chemicals (NaF and fluosilicates) in farm animals. Qualitative or quantitative determination of F in biological materials can be done by color reaction with zirconium-alizarin lake; presence of F yields a yellow color. The lake is added to the solution after decolorization of the latter by means of charcoal.

"Blastomogenic Action of Some Derivatives of 3,4'-ace-1,2-benzanthracene," L. M. Shabad, S. A. Buvaylo

"Byull Eksptl Biol i Med" Vol 21, 1946, pp 20-3

Subcutaneous administrations of 9,10-dimethyl-3,4'-ace-1,2-benzanthracene and the corresponding 9-methyl and 10-methyl derivatives were made in mice. Six of the 21 treated with 9,10-dimethyl derivative yielded sarcomas. The 9-methyl derivative gave nine sarcomas out of ten mice used (of partially known lineage) and similar results in graduated dosage tests using pure bred mice. Mice used in the 10-methyl derivative experiment died of other causes before diagnosis could be made. A large number of mice treated with the 9-methyl derivative also developed lung adenomas.

"Influence of Purity on the Toxicity of Trinitrotoluene," R. M. Sklyanskaya, F. I. Pozharskiy

"Farmakol i Toksikol" Vol 9, No 4, 1946, pp 61-4

Commercial TNT, rich in isomers, is more toxic to mice than pure TNT. Mortality rate is the same for crude and partly purified (Na_2SO_3-treated) TNT as for pure TNT when the dose is small but at higher dosages the mortality was highest for crude, followed by partly pure and pure TNT. Morphological changes such as liver necrosis were compared; they confirmed the evidence of mortality rates that pure TNT is inherently but only moderately toxic.

"Gastrointestinal Tract Burns by Corrosive Chemicals," A. I. Fel'dman

"Vestnik Oto-Rino-Laringol" Vol 8, No 4, 1946, pp 3-7

Symptomology and general treatment methods for cases of ingested corrosive chemicals such as acids or bases.

"Action of Gossypol on Animals (Dogs, Rabbits, Young Swine)," I. E. Mozgov

"Veterinariya" Vol 23, No 2/3, 1946, pp 38-42

Dogs given solid gossypol daily with their feed in doses of 1-3 mg per kg of body weight showed no clinical symptoms of poisoning, nor was any abnormality observed in dissecting the animal after 20 days; 5 mg caused signs of poisoning, and in solution (olive oil) a 3-mg dose caused inflammation and degeneration throughout the system. Effect was more pronounced on young swine, and somewhat less so on rabbits. Increasing the dose to 150-200 mg was reflected in localized reaction in 6-32 hours, followed by ulcerating and necrosis of the tissues, and disturbance of the heart functions. Gossypol is poisonous to the cell, vessel, and nerve, and the inflammatory process may last up to 50 days after administration or cause death.

"Comparative Pharmacological Action of Imidazoles Derived from Pyridine or Pyridine-pyrrolidine," B. L. Konson

"Farmakol i Toksikol" Vol 9, No 2, 1946, pp 3-9

Condensation products of α-halo ketones with α- and α'-amino nicotines were tested for pharmacological properties. The compounds were derivatives of 6-(N-methyl-α-pyrrolidine)pyrimidazole (I), namely its hydrochloride (II); Et 2-carboxylate (III); 2-carboxamide (IV); 2-phenyl derivative, hydrobromide (V); 2-phenyl derivative, hydrochloride (VI);

2-phenylpyrimidazole (VII); 3-phenylpyrimidazole-HCl (VIII); and 2-carboxy-3-phenylpyrimidazole-HCl (IX). Toxicities, as ratios to the toxicity of I to mice, are: III, 2; IV, 1/3; V, 1.5; VI, 2. Toxicity of VIII is about 1/3 that of VII and twice that of IX. As a depressant to the central nervous system VIII is more active than VII, but VI has higher spasmodic activity. Tolerated, toxic, and lethal doses were determined for each compound.

"Prevention of Mercury Poisoning in the Manufacture of Organic Mercury Compounds," N. P. Malyshev

"Gigiyena i Sanitariya" Vol 11, No 5, 1946, pp 20-3

In the preparation of organic Hg compounds, poisoning is due to the relatively high vapor pressure of Hg at the high temperature of the process. By introducing protective clothing, improving the ventilation, and increasing the mechanization it was possible to reduce the hazard. Removal of Hg adsorbed on walls and floors produced a drop of Hg concentration in the air, from 0.30-0.08 mg/cu m in 1943 to 0.12-traces in 1945. To remove Hg, the floors (Metlach tiles) were wetted with a solution of one part of $CaOCl_2$ in four parts H_2O, and after 2-3 hours (when all the Hg was converted into $HgCl_2$) were rinsed with water. Next, both floors and walls (oil paint) were washed with a 5-10% Na_2S solution, preferably with an excess 10% S_2, and after 24 hours, rinsed with soap and water. Hg was thus converted into HgS and removed in this form. Humidity and temperature do not affect the Hg concentration in the air of these plants.

"Toxicity of Sovcaine and Its Decrease Following Predosage with Glucose," A. Ya. Medvedev

"Farmakol i Toksikol" Vol 9, No 1, 1946, pp 38-42

Simultaneous administration of glucose (I) raises rather than lowers toxicity of sovcaine (II). In tests with dogs most effective predosage with I was 0.25 g/kg, given subcutaneously in 5 or 10% solution 30 minutes before injecting II. Toxicity of II is greatly lessened by this method. The lethal dose of II is not 0.0056 g/kg as has been reported; properly administered, II kills dogs at 0.02 g/kg and cats at 0.143 g/kg. Rapid intravenous injection of II in rabbits often causes sudden death by respiratory paralysis; slow injection of the same dose is safe. These experiments offer a basis for clinical experiments in the use of I to reduce the toxicity of II.

"Changes in Residual Nitrogen and Reserve Blood Alkalinity in Sheep and Their Prognostic Significance in Bis(2-chloro-ethyl) Sulfide Poisoning," P. E. Radkevich

"Farmakol i Toksikol" Vol 9, No 6, 1946, pp 52-3

Normal residual N content of sheep blood is not changed much by small oral doses (up to 1 cc) of $(ClC_2H_4)_2S$ but drops sharply after a lethal dose (2 cc). Hence, determinations of residual N offer criterion of severity and probable outcome of poisoning. Even a dose of 0.1-0.2 cc decreases reserve blood alkalinity; this offers another criterion for diagnosis and prognosis.

"Carbon Coefficient of Urine in Acute Aniline Poisoning," S. N. Sinitsyn

"Farmakol i Toksikol" Vol 9, No 2, 1946, pp 39-45

Total C in urine of test dogs and of patients with $PhNH_2$ (I) poisoning was determined by the Gomez method. Normally the C:N ratio (II) in urine is below 1, generally about 0.78 in dogs and 0.86 in man. Poisoning with I increases II, often to values above 1, probably by inhibiting oxybiotic processes. Tests were made with dogs of known average II on a prescribed low-protein diet. Poisoning was by subcutaneous injections of I; doses varied from 0.03 to 0.14 cc/kg. Patients with occupational I poisoning were placed on a low-protein diet before urine analysis.

"Mechanism of the Action of Methanol," A. T. Suprunov

"Farmakol i Toksikol" Vol 9, No 2, 1946, pp 49-51

Rabbits were given subcutaneous injections of MeOH in sublethal doses (12-14 ml/kg) and the effects on ascorbic acid (I), thiamine (II), and pyruvic acid (III) content were noted in liver, kidneys, heart, muscle, and brain. Dehydrogenation capacity was also determined. Averages from test rabbits and controls are shown in table. Oxidation of MeOH in tissues accompanies increased consumption of vitamin C and II in respiratory processes. Depletion of vitamin reserves disturbs the oxidation-reduction cycle, leading to anoxemia. In view of the active participation of II and vitamin PP in intracellular oxidation, vitamin therapy utilizing I, II, nicotinic acid, and riboflavin is recommended in acute MeOH poisoning.

"Dissociation Curves of Oxyhemoglobin in Toxic Fever," A. M. Charnyy, S. E. Krasovitskaya, F. E. Syrkina

"Farmakol i Toksikol" Vol 9, No 4, 1946, pp 49-53

In toxic fever induced by dinitrophenol (I) the dissociation curve of oxyhemoglobin (II) shifts up and leftward from its normal position, not because of acidosis or hyperthermy, but apparently in relation to erythrocyte changes. It is significant that the curve did not shift downward even when the temperature rose to 44.8 degrees. Test animals (dogs) received

I (30 mg/kg) in alcohol solution by subcutaneous injection. This dose effects a temperature rise of about one degree per hour and a corresponding shift in the dissociation curve of II.

"Influence of High and Low Temperatures on Stability of Lobeline Solutions," P. P. Saksonov

"Farmakol i Toksikol" Vol 9, No 3, 1946, pp 25-8

Synthetic lobeline-HCl (I) in 1% solution was frozen 15 days, then boiled 90 minutes. It retained its activity as a respiratory stimulant and its pressor effect in healthy human subjects, as well as its toxicity to rats. These results demonstrate the stability of I, but chemical and pharmacological studies of purity standards and physiological action are still needed.

"Pharmacology of Eremosparton Aphyllum," A. A. Lyubushin, Kazakh Republic Psychiatric Hospital, Kzyl-Orda

"Farmakol i Toksikol" Vol 9, No 2, 1946, p 30

Eremosparton aphyllum is a poisonous plant growing in Kazakhstan. Parts above the roots are rich in alkaloids. The extract (I) (1:10) in Ringer solution causes rigidity, motor nerve stimulation, and intensified reflexes in frogs. Dose of 100 g/kg kills frogs in 1-2 hours by paralysing the central nervous system, though muscles and peripheral nerves remain excitable. Isolated frog heart, perfused with more dilute I (1:100) shows increased amplitude without change of rhythm; at 1:20 or 1:10 there is a negative chronotropic action, with decreased amplitude and a cardiac failure in diastole. Intravenous injection of I (1:10) in decerebrated dogs slows respiration, lowers blood pressure, and raises pulse rate. In five tests with human digits, isolated by the Kravkov method, perfusion with I (1:1,000) caused a prolonged vasoconstrictor effect. Thus, it appears that lowered blood pressure in dogs is due to selective action on the vasomotor centers, not to cardiac depression nor to peripheral action on the vessel walls.

"A Flare-up of Food Toxico-infections," D. A. Drobinskiy, G. Ya. Zmeyev

"Zhur Mikrobiol, Epidemiol, i Immunobiol" 1946, No 1/2, pp 23-6

Number of cases of food poisoning by meat are described, and it is shown that the toxic effects were most probably caused by the Gaertner bacillus.

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"Influence of Carbon Monoxide on Higher Nervous Activity," Yuriy P. Frolov

"American Review of Soviet Medicine" Vol 4, 1946, pp 90-1

Various degrees of CO poisoning can be observed by giving subthreshold doses of CO which will accustom an organism to the poison. Severely poisoned newborn white rats and dogs whose brain cortex is not yet fully developed are not sensitive or show little reaction to CO in large concentrations. Although the absorption of CO by the blood of both adult and young animals was the same and the respiratory functions in both cases were reduced to 70-80%, absence of a developed brain cortex in the newborn prevented death, even with strong concentrations. In experiments on dogs with different forms of higher nervous activity behavior, single and repeated CO poisonings showed clear-cut functional changes in the major cortical processes of excitation and inhibition, while the lower parts of the brain and the pons varolii were unaffected. In particular, the unconditioned salivary reflexes remained unchanged; there was a considerable decrease in the conditioned salivary reflexes and a weakening of active internal inhibition. A weakening of the conditioned motor reflexes indicated changes in the motor zone of the cortex. Disturbances of peripheral type were observed, especially eczema.

"Experiments in Feeding Hogs with Toxic Fodder," A. Ya. Lukin, M. G. Berlin

"Veterinariya" Vol 23, No 1, 1946, pp 36-7

In connection with the attack of human septic angina through consumption of millet left to winter uncut in the field, feeding experiments were conducted on animals. Hogs were not harmed by the grain which was toxic to man. Work on feeding horses was only preliminary and not conclusive.

"Nonoccupational Poisoning with Tetraethyl Lead," E. N. Marchenko, G. A. Belikis

"Gigiyena i Sanitariya" Vol 11, No 9, 1946, pp 17-23

Widespread use of Et_4Pb in petroleum fuels in the past few years was responsible, indirectly, for a large number of poisonings of individuals and groups. Poisoning was caused by its use as a fuel for heating and cooking stoves, lighting, kindling, as a solvent, spray, or by the use of insufficiently cleaned drums or tank cars. Greater percentage of poisoning cases was due to inhalation of vapors or combustion gases, but greater percentage of fatalities was caused by its entry by mouth; of these, 19% of the fatalities

were caused by food and 72% by drink. Recommendations are given for stricter control and better education of the personnel.

"Mechanism of Action of Arsine: III, Sodium Nitrite Prophylaxis against Arsine Poisoning," V. M. Rozhkov, S. A. Caherov

"Farmakol i Toksikol" Vol 9, No 3, 1946, pp 47-51

When dogs are given $NaNO_2$ (0.02 g/kg), blood methemoglobin (I) increases 30-35% in 2 hours. If the $NaNO_2$ is followed by exposure to AsH_3 for an hour, I decreases instead of increasing. The minimum lethal dose of AsH_3 is 15 minutes exposure to 0.6 mg/l. Prophylactic use of $NaNO_2$ within an hour before poisoning with AsH_3 strongly diminishes rate and intensity of hemolysis, which is the usual cause of death.

"Mechanism of Toxic Emphysema in Very Young Animals," V. D. Rozanova

"Farmakol i Toksikol" Vol 8, No 1, 1945, pp 8-11

Irritation of tracheal or bronchial mucosa with 7-20% H_2SO_4 produces inflammatory emphysema (I) in rabbits a few hours old, even in tracheas cut transversely at the base. Intravenous injection of 10% chloramine solution (II) causes I in newborn rabbits, but not in embryos before severing the umbilical cord. The dose of II ranged from 1 to 5 cc/kg for rabbit embryos down to 0.5 cc/kg for dogs and 0.1 cc/kg for rabbits.

"Comparative Toxicity of Benzylaniline and N-methyldiphenylamine," N. A. Sazonova

"Farmakol i Toksikol" Vol 8, No 2, 1945, p 50

Tested on mice and rabbits, $PhNHCH_2Ph$ (I) is more toxic than Ph_2NMe (II) in peroral, subcutaneous, or respiratory dosage. Applied directly to rabbit skin, both are slightly toxic, II a little more than I. Tests were made to ascertain possible hazards in using I or II as insecticides.

"Theory of Acute Hydrogen Sulfide Poisoning," I. Olyvin, A. I. Gunina, V. I. Olyvin

"Byull Eksptl Biol i Med" Vol 19, No 6, 1945, pp 44-7

Pathogenesis of H_2S poisoning is not identical to that of HCN, as postulated by Rohdenacker and others, since tissue respiration is not as markedly affected by H_2S. Appearance of a short-lived increase of O in the venous

blood during acute H_2S poisoning is not the result of suppression of tissue respiration, but is due to an increase of blood pressure; same effect is produced by adrenaline. Direct action of the H_2S on the central nervous system is the cause of death.

"Influence of Chinese Schisandra Fruits on Spinal Centers," F. E. Pozdyankov

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 15-19

Finely powdered schisandra fruits, 0.5-2 g/kg per os or by direct introduction into the stomach, stimulate the spinal reflexes of posterior extremities in dogs after low total chordotomy. Motor activity and general behavior are not significantly changed. Probably similar neurodynamic changes in anterior parts of the body require a different technique, e. g., conditioned reflexes, chronaxia, ergographic tests, or the like. Larger dose causes hyperkinesis, emotional stimulation, heightened posterior spinal reflexes, and retarded urination and defecation in 1-2 hours. Smaller doses take 4-6 hours; symptoms last 4-20 hours. Schisandra fruits appear to be suitable for use in stimulants for the central nervous system.

"Biochemical Properties of Toxic Millet," V. Kretovich, N. Sosedov, Z. Skripkina, V. Shvetsova, Moscow Inst Cereal Research

"Biokhimiya" Vol 10, 1945, pp 279-84

Grain is often found to be toxic as a food if it has lain all winter in the field, covered with snow. Toxic millets differ from normal grain in having a higher content of nonprotein and amino N, and a lower activity of oxidizing enzymes. Dextrin formation by amylase, as determined by Wohlgemuth's method, is twice as high in toxic millet.

"Influence of Some Analeptics on the Sechenov Inhibition," V. V. Zakusov

"Farmakol i Toksikol" Vol 8, No 5, 1945, pp 3-6

Pentamethylenetetrazole (corazole, I), coramine (II), $PhC(CH_2)Me_2$ sulfate (phenamine, III), and strychnine (IV) may weaken, but do not halt, the Sechenov inhibition of cerebral stimuli in the central nervous system by crystalline NaCl. Neither does crystalline NaCl halt convulsions caused by I, II, III, or IV. Inactivity of I, II, III, and IV toward the Sechenov inhibition is attributed to excessive stimulation of the thalamic centers of pain sensitivity. Tests were made with frogs using 50-100% of the convulsion-inducing dose of I, II or IV. The dose of III, which does not cause convulsions in frogs, was 4% of the lethal dose.

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"Effect of Aniline on Tissue Respiration," Z. A. Il'yina

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 47-8

By inhibiting respiratory oxidation processes, $PhNH_2$ retards tissue respiration in the liver, kidneys, spleen, and diaphragm. Tests were made with rats, exposed 1-3 hours to air containing $PhNH_2$ vapor.

"Blood Glutathione in Experimental Chronic Aniline Poisoning," N. S. Pravdin, S. D. Shakhnovskaya

"Farmakol i Toksikol," Vol 8, No 4, 1945, pp 49-53

In chronic poisoning of dogs by exposure daily to $PhNH_2$ vapor, total glutathione (I) changes only slightly at first although the ratio of reduced and oxidized I (GSH and GSSG) varies. The ratio GSH:GSSG first falls, then rises; finally total GSSG increases both by oxidizing GSH and by increase in total I. These experiments are significant in relation to the mechanism of $PhNH_2$ poisoning.

"Pharmacological Study of Crystalline Coronillin," P. I. Oniteev

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 3-6

Coronillin (I) was prepared from crushed (Coronilla varia L.) seeds and tested on isolated frog heart, rabbit intestine, and rabbit uterus in concentrations of 0.1-40 ppm. In general, I acts like other cardiac glycosides. Lethal dose for cats is 0.22 mg/kg. At 10 ppm it causes systolic stoppage of isolated frog heart. Crystalline I is about five times as active as amorphous I; so is strophanthin K. In dogs (0.2 mg/kg intravenously) blood pressure rises; cardiac rhythm is first sharply slowed, then accelerated; amplitude first falls off sharply, then rises. Strips of isolated smooth muscle (rabbit uterus or intestine), treated with I at 2 ppm, showed heightened tonus and cessation of contraction. Tonus returned to normal when I was washed out. Use of I from Soviet sources as a cardiac glycoside is recommended.

"Toxicology of Ethyl Bromide," A. B. Reznikov

"Farmakol i Toksikol" Vol 8, No 3, 1945, pp 58-9

Clinical studies of workers poisoned by EtBr are reported. The Br content of the blood is a useful diagnostic test.

"Biochemical Changes in Blood and Urine in Severe Acute Aniline Poisoning," P. A. Rozenberg

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 23-4

In an unsuccessful suicide attempt by taking about 100 g of aniline oil and alcohol, the changes in blood and urine were more pronounced than in ordinary occupational poisoning, as shown by the following comparison: Sharper rise of methemoglobin (I); slower disappearance of I from the blood and of para-aminophenol from the urine; more bilirubin; severe hemoglobinuria; severe instead of very mild hemolysis as shown by hemoglobin drop and erythrocyte count; sharper drop in glutathione (II), reduced and oxidized, and in Gabbe index; higher early free phenol content in urine; and lower content of combined phenols.

"Biochemical Changes in Blood under Chronic Action of Aniline and Other Amino- and Nitro-benzene Derivatives," G. V. Degwiz, P. A. Rozenberg

"Farmakol i Toksikol" Vol 8, No 5, 1945, pp 40-3

Clinical observations on 27 patients with 3 months to 22 years of occupational exposure in the Dorkhim chemical works showed that blood phenols, urine phenols, hemoglobin, erythrocyte count, blood glutathione, and serum bilirubin fluctuated mostly within normal limits except that urine phenols tended to be high. Ratio of combined to free phenols varied from 0.1 to about 5. Changes in blood bilirubin gave evidence of hemolysis. Though glutathione content varied considerably, glutathione index remained within normal limits since high glutathione and high erythrocyte count went together.

"Toxicology of Hydrazoic Acid," N. S. Pravdin, S. B. Shakhnovskaya

"Farmakol i Toksikol" Vol 8, No 5, 1945, pp 50-4

Mortality of mice exposed to HN_3 vapor at various concentrations (in mg/liter) was found to be: 0.01, none; 0.02, 100% in 3 hours; 0.1, 100% in 2 hours. At 0.2, acute poisoning was apparent in 5-10 minutes. In subacute poisoning of rabbits, vapor concentration 0.01-0.04, time 2 hours daily, death ensued in 15-42 days after 13-28% weight loss. Central nervous system was affected; toxic effects include depression of the vasomotor center and respiratory paralysis.

"Pathomorphological Changes of Internal Organs in Poisoning with Dichloroethane through the Alimentary Tract," F. F. Bryshin

"Farmakol i Toksikol" Vol 8, No 5, 1945, pp 43-9

Death from acute $C_2H_4Cl_2$ (I) poisoning follows paralysis of the central nervous system. Autopsies on four young men who took I by mistake attest the high toxicity of I, which is one of the most active of the aliphatic halide narcotics. Medicolegal decisions should be based on the fullest possible information from clinical records, autopsy (including histological examination of internal organs), and chemical tests.

"Anabasine Sulfate Poisoning," N. S. Zhelezhyanskaya

"Gigiyena i Sanitariya" No 6, 1945, pp 20-3

In 26 cases of accidental poisoning with anabasine sulfate (admixture to food), 20 patients showed severe symptoms, which developed soon after the food intake. The irritation of the mucous membranes of the mouth and the stomach, caused by doses of 20-52 mg, lasted a long time, but there were no pathological changes in the gastrointestinal tract.

"Changes in Alkali Reserve and pH in the Blood in Acute Poisoning with Amino- and Nitro-benzene Derivatives," N. M. Israilskaya, P. A. Rozenberg

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 53-4

From data on 19 dogs poisoned with $PhNH_2$, and clinical studies of 10 patients poisoned with $PhNH_2$, $PhNMe_2$, $PhNO_2$, or para-nitraniline, it appears that alkali reserve is decreased. Generally, the loss goes beyond the buffering capacity of the blood, so that acid accumulates and pH decreases. The effect is attributed to incomplete oxidation, since these poisons inhibit respiratory oxidation.

"Biochemical Changes in Blood and Urine in Acute Poisoning with Aniline and Other Amino and Nitro Derivatives of Benzene," G. V. Dervis, P. A. Rozenberg

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 19-23

Clinical studies of 30 victims of acute poisoning by $PhNH_2$, $PhNO_2$, or meta- or para-nitraniline showed that methemoglobin disappears from the blood, and para-aminophenol from the urine, about the second day. First blood test shows glutathione near its upper normal limit. Gibbe index is often increased, but drops back to normal. Bilirubin rises in the first few days but returns to normal with convalescence. Combined phenols in blood and urine are increased, and part of the increase persists for a long time.

"Detoxifying Functions of the Liver and Other Organs in Dinitrobenzene Poisoning," N. L. Beloborodova

"Farmakol i Toksikol" Vol 8, No 4, 1945, pp 32-6

In rabbits poisoned with meta-$C_6H_4(NO_2)_2$ (I), given in vegetable oil per os or subcutaneously, in dose of 0.05-0.1 g/kg, the liver quickly removes I from the blood and converts it. Muscles and kidneys have the same power, less strongly; but meta-nitroaniline (II), one of the conversion products, is found in kidneys and muscles, not in the liver. Kidneys assist in the conversion, and take I and II out of the blood for excretion. Muscles also participate in converting I to II.

"Antagonism between Para-Aminobenzoic Acid and Sulfanilamide," N. I. Efimov

"Farmakol i Toksikol" Vol 8, No 5, 1945, pp 28-9

Sulfanilamide (I) at 10-10,000 ppm and para-$H_2NC_6H_4$-COOH (II) at 10-200 ppm were tested on isolated frog hearts. Neither rhythm nor amplitude was changed by II, nor by I at 10-40 ppm. At 100 ppm I begins to increase amplitude, but not to accelerate rhythm. At 5,000 ppm I stops the frog heart; at 200 ppm II offsets the depressant effect of 10,000 ppm of I. Referred to the effect of K and Ca ions, I is K-type and II is Ca-type in action.

"Poisoning with Glysantin and Antifreeze," S. Ya. Arbuzov

"Farmakol i Toksikol" Vol 8, No 3, 1945, pp 55-8

The German glysantine (nearly identical to Prestone) sometimes contains denaturants such as mercaptans. Clinical records are presented for eight cases of poisoning. First stage of poisoning is euphoria, lasting about a day. The second or toxic stage, with anoxemia, lasts 2-3 days. Oxygen therapy is beneficial along with other remedies such as glucose, strophanthin, and a variety of analeptics.

"Effects of Benzene and Gasoline on the Organism at Low Atmospheric Pressures," N. S. Shvarsalov

"Farmakol i Toksikol" Vol 8, No 3, 1945, pp 51-5

Experiments with mice poisoned by C_6H_6 vapor (I) at pressures corresponding to sea level, 2,000 meters (396 mm Hg), and 4,000 meters (462 mm Hg) altitude show some increase in toxicity under prolonged exposure at 596 mm and a somewhat greater increase at 462 mm, with earlier death. With aviation gasoline

B-70 (II) there was earlier loss of muscle coordination than with I, sharper decrease or cessation of excitation periods, and earlier narcosis. Partial pressure of O_2 is a factor in toxicity, both for I and for II.

"Clinical Studies of Poisoning by Organic Mercurials (Diethylmercury Phosphate and Diethylmercury)," E. A. Drogichina, S. D. Gurzo

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 41-2

From eight cases of chronic poisoning by vapors of Et_2Hg and its phosphate it appears that the encephalopathic effects are different from those of Hg poisoning. There are distinct psychic symptoms. Hemoglobin count dropped 44-50%, with some lymphocytosis and monocytosis. The Hg content of the urine reached 0.6 mg/liter. Stomatitis, quickly progressing to ulcerating gingivitis, was a symptom.

"Pathogenesis of Liver Lesions in Trinitrotoluene Poisoning," D. E. Al'pern, Kurlyandskaya, Romashevskaya, Kisel, Evgenova

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 42-44

Tests with dogs and rabbits, and with tissues in vitro, indicate that the liver is especially active in chemical conversion of TNT; the kidneys and some other organs also participate. Among the toxic effects are swelling and proliferation of cells in the reticuloendothelial system, with degeneration of liver cells. Peroral and subcutaneous dosage give identical effects.

"Action of Merendera (Merendera robusta Bge) Poison on Some Rodents"

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 54-57

Merendera is not a generally applicable rat poison. It is toxic to white mice and house mice, but not to voles. Eating colchicine-bearing plants does not decrease the susceptibility of rodents to merendera. Plants yielding colchicine are not suitable for rat control.

"Influence of Acute and Chronic Carbon Monoxide Poisoning on the Activity of Higher Nerves in Animals," L. S. Gorshaleva

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 47-51

Chronic poisoning by CO in doses too small to be detected by blood tests can be diagnosed by the method of conditioned reflexes. There is evidence of direct action on the central nervous system, with sharper

reaction than in mild single-dose poisoning. Earliest effects in chronic CO poisoning include cortical changes in the upper parts of the central nervous system. Trophic changes in the skin also occur.

"Acute Water Hemlock Poisoning," M. M. Petrachkov, S. F. Dement'yeva

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 57-61

The active principle of water hemlock (Cicuta virrosa) is a spasmodic with high toxicity to the central nervous system and a pronounced pressor effect. It accelerates respiration, especially during paroxysms, but finally causes respiratory paralysis and death. It is a local stimulant to certain internal organs and tissues. The poison is stable at boiling temperature and durable in storage even if exposed to air.

"Effects of Sodium Fluoride on the Leucocyte Picture in Man," I. N. Davydov

"Farmakol i Toksikol" Vol 7, No 5, 1944, pp 37-41

Toxic doses of NaF in man cause prolonged leucopenia, with relative and absolute neutropenia and relative lymphocytosis. The absolute number of lymphocytes decreases somewhat. This reaction apparently reflects the leucolytic action of NaF and could be used in diagnosis of fluoride poisoning. The same doses of NaF cause no change in the erythrocyte picture. Curve charts show the effects of NaF on leucocyte, neutrophil, and monocyte counts.

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